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Search Results -

Terms	Documents
Dog with (P-glycoprotein or MDR1)	7

Database: US Patents Full-Text Database; US Pre-Grant Publication Full-Text Database; JPO Abstracts Database; EPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Search: L1

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DATE: Tuesday, December 10, 2002 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=ADJ			
L1	Dog with (P-glycoprotein or MDR1)	7	L1

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Search Results - Record(s) 1 through 7 of 7 returned.

☐ 1. Document ID: US 20020177147 A1

L1: Entry 1 of 7 File: PGPB Nov 28, 2002

PGPUB-DOCUMENT-NUMBER: 20020177147
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020177147 A1

TITLE: Mdr1 variants and methods for their use

PUBLICATION-DATE: November 28, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Mealey, Katrina L.	Pullman	WA	US	
Bentjen, Steven A.	Troy	ID	US	

US-CL-CURRENT: 435/6

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

☐ 2. Document ID: US 6171786 B1

L1: Entry 2 of 7 File: USPT Jan 9, 2001

US-PAT-NO: 6171786
DOCUMENT-IDENTIFIER: US 6171786 B1

TITLE: Methods for preventing multidrug resistance in cancer cells

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

☐ 3. Document ID: US 5972598 A

L1: Entry 3 of 7 File: USPT Oct 26, 1999

US-PAT-NO: 5972598
DOCUMENT-IDENTIFIER: US 5972598 A

TITLE: Methods for preventing multidrug resistance in cancer cells

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

☐ 4. Document ID: US 5928637 A

L1: Entry 4 of 7 File: USPT Jul 27, 1999

US-PAT-NO: 5928637
DOCUMENT-IDENTIFIER: US 5928637 A

TITLE: Methods of inducing multidrug resistance using human MDR1 cDNA

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

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☐ 5. Document ID: US 5851819 A

L1: Entry 5 of 7 File: USPT Dec 22, 1998

US-PAT-NO: 5851819
DOCUMENT-IDENTIFIER: US 5851819 A

TITLE: Vectors carrying MDR1 cDNA which confer multidrug resistance on transduced cells

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

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☐ 6. Document ID: US 5849998 A

L1: Entry 6 of 7 File: USPT Dec 15, 1998

US-PAT-NO: 5849998
DOCUMENT-IDENTIFIER: US 5849998 A

TITLE: Transgenic animals expressing a multidrug resistance cDNA

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

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☐ 7. Document ID: WO 200123540 A2 EP 1220911 A2 AU 200077327 A

L1: Entry 7 of 7 File: DWPI Apr 5, 2001

DERWENT-ACC-NO: 2001-235373
DERWENT-WEEK: 200253

TITLE: New dog P-glycoproteins (PGP) and their encoding nucleic acids, useful for determining the bioavailability of drugs and for screening for dog PGP inhibitors

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

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Terms	Documents
Dog with (P-glycoprotein or MDR1)	7

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     INDEX 'ADISALERTS, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, AQUASCI,
     BIOBUSINESS, BIOCOMMERCE, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA,
     CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DDFB,
     DDFU, DGENE, DRUGB, DRUGLAUNCH, DRUGMONOG2, ...' ENTERED AT 08:31:24 ON
     10 DEC 2002
                SEA (P-GLYCOPROTEIN OR PGP OR MDR1) AND (DOG OR CANINE)
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                 4   FILE ADISALERTS
                 9   FILE ADISINSIGHT
                 5   FILE AGRICOLA
                 2   FILE BIOBUSINESS
               122   FILE BIOSIS
                 3   FILE BIOTECHABS
                 3   FILE BIOTECHDS
                26   FILE BIOTECHNO
                 9   FILE CABA
                61   FILE CANCERLIT
                88   FILE CAPLUS
                 3   FILE CONFSCI
                 1   FILE CROPU
                 1   FILE DDFB
               153   FILE DDFU
                46   FILE DGENE
                 1   FILE DRUGB
                 4   FILE DRUGNL
               164   FILE DRUGU
                 8   FILE DRUGUPDATES
                 4   FILE EMBAL
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                39   FILE ESBIOBASE
                 3   FILE FEDRIP
                 9   FILE GENBANK
                 3   FILE IFIPAT
                 7   FILE JICST-EPLUS
                17   FILE LIFESCI
               106   FILE MEDLINE
                42   FILE PASCAL
                 7   FILE PHAR
                67   FILE PROMT
               119   FILE SCISEARCH
                72   FILE TOXCENTER
               427   FILE USPATFULL
                 9   FILE USPAT2
               110   FILE VETU
                 5   FILE WPIDS
                 5   FILE WPINDEX
L1               QUE (P-GLYCOPROTEIN OR PGP OR MDR1) AND (DOG OR CANINE)
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     FILE 'DRUGU, BIOSIS, SCISEARCH, VETU, MEDLINE, CAPLUS, EMBASE, TOXCENTER'
     ENTERED AT 08:34:02 ON 10 DEC 2002
L2           854 S L1
L3           469 DUP REM L2 (385 DUPLICATES REMOVED)
L4            15 S L3 AND (VARIANT OR MUTANT OR ALLEL?)
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=> d l4 ibib ab 1-15

L4 ANSWER 1 OF 15 DRUGU COPYRIGHT 2002 THOMSON DERWENT
ACCESSION NUMBER: 2001-24569 DRUGU P
TITLE: Increased functional cell surface expression of CFTR and deltaF508-CFTR by the anthracycline doxorubicin.
AUTHOR: Maitra R; Shaw C M; Stanton B A; Hamilton J W
CORPORATE SOURCE: Dartmouth-Med.Sch.
LOCATION: Hanover, N.H., USA
SOURCE: Am.J.Physiol. (280, No. 5, Pt. 1, C1031-C1037, 2001) 4 Fig. 25 Ref.
CODEN: AJPHAP ISSN: 0002-9513
AVAIL. OF DOC.: Dept. of Pharmacology and Toxicology, Dartmouth Medical School, 7650 Remsen, Hanover, NH 03755-3835, U.S.A. (J.W.H.). (e-mail: josh.hamilton@dartmouth.edu).
LANGUAGE: English
DOCUMENT TYPE: Journal
FIELD AVAIL.: AB; LA; CT
FILE SEGMENT: Literature
AB A single, non-cytotoxic dose of doxorubicin (Dox) enhanced cystic fibrosis transmembrane conductance regulator (CFTR) protein expression at the cell surface in human colon adenocarcinoma T84 epithelial cells and increased CFTR-mediated chloride permeability but had little or no effect on CFTR mRNA expression. Dox increased CFTR-mediated chloride secretion and also green fluorescent protein (GFP) tagged delta-F508-CFTR expression in stably transfected Madin-Darby **canine** kidney (MDCK) cells expressing the **mutant** GFP-delta-F508-CFTR. Results suggest that anthracycline analogs may be useful for the treatment of cystic fibrosis.

L4 ANSWER 2 OF 15 DRUGU COPYRIGHT 2002 THOMSON DERWENT
ACCESSION NUMBER: 2001-10045 DRUGU T
TITLE: Hematopoietic stem cell transplants (HSCT) from unrelated donors using low dose TBI, fludarabine and a combination of cyclosporine and mycophenolate mofetil.
AUTHOR: Niederwiesser D W; McSweeney P; Wolff D; Hegenbart U; Mantovani L; Pnisch W; Deininger M; Edelmann J; Kamprath F; Blume K G
CORPORATE SOURCE: Univ.Colorado; Univ.Stanford; Fred-Hutchinson-Cancer-Res.Cent.
LOCATION: Leipzig, Ger.; Denver, Colo., Stanford, Cal.; Seattle, Wash., USA
SOURCE: ; Proc.Am.Soc.Clin.Oncol. (19, 36 Meet., 47a, 2000) CODEN: ; 7790
AVAIL. OF DOC.: Division of Hematology/Oncology, University of Leipzig, Leipzig, Germany. (12 authors).
LANGUAGE: English
DOCUMENT TYPE: Journal
FIELD AVAIL.: AB; LA; CT
FILE SEGMENT: Literature
AB Based on studies in a **canine** model, an approach for hematopoietic stem cell transplants (HSCT) from unrelated donors was developed using 200 cGy total body irradiation (TBI) before and a combination of mycophenolate mofetil (MMF) and ciclosporin (CSP) after transplant. This approach has been successfully applied to over 60 patients with hematological malignancies treated by HLA-identical sibling transplants, although there was a 15% nonfatal rejection rate. Fludarabine (FLU) was added to decrease the risk of rejection. Early results for 18 patients confirmed that allogenic HSCT is possible even in older patients using HLA-matched or 1 HLA antigen mismatched donors. Graft rejection was rare, while graft vs. host disease (GvHD) was seen in 50% of patients. (conference abstract: 36th Annual Meeting of the American Society of Clinical Oncology, New Orleans, Louisiana, USA,

2000).

L4 ANSWER 3 OF 15 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2002:269366 BIOSIS
DOCUMENT NUMBER: PREV200200269366
TITLE: Frequency of the **mutant MDR1** **allele** associated with ivermectin sensitivity in a sample population of Collies from the northwestern United States.
AUTHOR(S): Mealey, Katrina L. (1); Bentjen, Steven A.; Waiting, Denise K.
CORPORATE SOURCE: (1) Department of Veterinary Clinical Sciences, Washington State University, Pullman, WA, 99164-6610 USA
SOURCE: American Journal of Veterinary Research, (April, 2002) Vol. 63, No. 4, pp. 479-481. print.
ISSN: 0002-9645.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Objective: To determine the frequency of the **MDR1** gene mutation (polymorphism) associated with ivermectin sensitivity in a sample population of Collies in Washington and Idaho. Animals: 40 healthy client-owned Collies. Procedure: A blood sample (8 ml) was collected from each **dog** and used for RNA extraction. Reverse transcriptase was used to generate **MDR1** cDNA. Polymerase chain reaction (PCR) primers were designed to amplify a 1,061-base pair region of the **MDR1** gene. The PCR products were sequenced to determine whether the Collies had 0, 1, or 2 **mutant alleles**. Pedigrees of some **dogs** were available for analysis to determine relatedness of affected **dogs**. Results: Of the 40 Collies, 9 (22%) were homozygous for the normal **allele** (normal), 17 (42%) were heterozygous (carrier), and 14 (35%) were homozygous for the **mutant allele** (affected). Pedigree analysis revealed that some, but not all, affected **dogs** were related to each other within the 4 most recent generations. Conclusions and Clinical Relevance: A high percentage of a sample population of Collies in Washington and Idaho are affected or carriers of the **mutant MDR1** **allele** associated with ivermectin sensitivity. A similar frequency of this mutation may be detected in **dogs** from other geographic areas. Pharmacologic treatment with ivermectin, loperamide, vincristine, and other drugs that are substrates of **P-glycoprotein**, the **MDR1** gene product, may result in neurologic toxicosis in a high percentage of Collies.

L4 ANSWER 4 OF 15 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2002:237426 BIOSIS
DOCUMENT NUMBER: PREV200200237426
TITLE: Role of glutathione conjugate efflux in cellular protection against benzo(a)pyrene-7,8-diol-9,10-epoxide-induced DNA damage.
AUTHOR(S): Srivastava, Sanjay K. (1); Watkins, Simon C.; Schuetz, Erin; Singh, Shivendra V.
CORPORATE SOURCE: (1) University of Pittsburgh, 3550 Terrace Street, S-845 Scaife Hall, Pittsburgh, PA, 15261 USA
SOURCE: Molecular Carcinogenesis, (March, 2002) Vol. 33, No. 3, pp. 156-162. http://www.interscience.wiley.com/jpages/0899-1987/. print.
ISSN: 0899-1987.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Glutathione (GSH) conjugation of (+)-anti-benzo(a)pyrene-7,8-diol-9,10-epoxide ((+)-anti-BPDE), the activated metabolite of benzo(a)pyrene, is believed to be an important mechanism in detoxification of this environmental and dietary carcinogen. Here, we demonstrate that the intracellular accumulation of GSH conjugate of (+)-anti-BPDE (BPD-SG)

caused a statistically significant increase in (+)-anti-BPDE-induced DNA adduction. The relationship between intracellular accumulation of BPD-SG and (+)-anti-BPDE-induced DNA adduction was studied using a **canine** kidney epithelial cell line (MDCKII) and its **variants** overexpressing multidrug resistance transporter (**MDR1**) or canalicular multispecific organic anion transporter (cMOAT; also known as multidrug resistance protein 2). **MDR1** and cMOAT are implicated in ATP-dependent efflux of anticancer drugs or GSH-xenobiotic conjugates, or both. The GST activity toward (+)-anti-BPDE in parental MDCKII cells did not differ from that in subline overexpressing **MDR1** (MDCKII-**MDR1**) or cMOAT (MDCKII-cMOAT). Intracellular accumulation of BPD-SG, after a 5- or 10-min incubation with 1 muM (+)-anti-BPDE, was significantly higher in parental (41- to 67-fold) and MDCK II-**MDR1** cells (31- to 43-fold) than in the MDCKII-cMOAT cells. Interestingly, the levels of DNA adducts of (+)-anti-BPDE, after a 30-min incubation with 0.1 or 0.5 muM (3H)(+)-anti-BPDE, were significantly higher (about 2.1- and 1.7-fold, respectively) in parental cells than in the MDCKII-cMOAT cells. The results of the present study indicate that in addition to GSH conjugation, the efflux of BPD-SG may be essential for cellular protection against (+)-anti-BPDE-induced DNA damage.

L4 ANSWER 5 OF 15 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 2001:331917 SCISEARCH
THE GENUINE ARTICLE: 424FF
TITLE: Increased functional cell surface expression of CFTR and Delta F508-CFTR by the anthracycline doxorubicin
AUTHOR: Maitra R; Shaw C M; Stanton B A; Hamilton J W (Reprint)
CORPORATE SOURCE: Dartmouth Coll, Sch Med, Dept Pharmacol & Toxicol, 7650 Remsen, Hanover, NH 03755 USA (Reprint); Dartmouth Coll, Sch Med, Dept Pharmacol & Toxicol, Hanover, NH 03755 USA; Dartmouth Coll, Sch Med, Dept Physiol, Hanover, NH 03755 USA
COUNTRY OF AUTHOR: USA
SOURCE: AMERICAN JOURNAL OF PHYSIOLOGY-CELL PHYSIOLOGY, (MAY 2001) Vol. 280, No. 5, pp. C1031-C1037.
Publisher: AMER PHYSIOLOGICAL SOC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814 USA.
ISSN: 0363-6143.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 26
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Cystic fibrosis (CF) is a disease that is caused by mutations within the cystic fibrosis transmembrane conductance regulator (CFTR) gene. The most common mutation, Delta F508, accounts for 70% of all CF **alleles** and results in a protein that is defective in folding and trafficking to the cell surface. However, Delta F508-CFTR is functional when properly localized. We report that a single, noncytotoxic dose of the anthracycline doxorubicin (Dox, 0.25 muM) significantly increased total cellular CFTR protein expression, cell surface CFTR protein expression, and CFTR-associated chloride secretion in cultured T84 epithelial cells. Dox treatment also increased Delta F508-CFTR cell surface expression and Delta F508-CFTR-associated chloride secretion in stably transfected Madin-Darby **canine** kidney cells. These results suggest that anthracycline analogs may be useful for the clinical treatment of CF.

L4 ANSWER 6 OF 15 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 2000:608576 SCISEARCH
THE GENUINE ARTICLE: 340YL
TITLE: MDR3 **P-glycoprotein**, a phosphatidylcholine translocase, transports several cytotoxic drugs and directly interacts with drugs as judged by interference with nucleotide trapping
AUTHOR: Smith A J; vanHelvoort A; vanMeer G; Szabo K; Welker E;

Szakacs G; Varadi A; Sarkadi B; Borst P (Reprint)

CORPORATE SOURCE: NETHERLANDS CANC INST, DIV MOL BIOL, PLESMANLAAN 121, NL-1066 CX AMSTERDAM, NETHERLANDS (Reprint); NETHERLANDS CANC INST, DIV MOL BIOL, NL-1066 CX AMSTERDAM, NETHERLANDS; NETHERLANDS CANC INST, CTR BIOMED GENET, NL-1066 CX AMSTERDAM, NETHERLANDS; UNIV AMSTERDAM, ACAD MED CTR, CELL BIOL & HISTOL LAB, NL-1105 AZ AMSTERDAM, NETHERLANDS; HUNGARIAN ACAD SCI, MEMBRANE RES GRP, NATL INST HAEMATOL & IMMUNOL, H-1113 BUDAPEST, HUNGARY; HUNGARIAN ACAD SCI, BIOL RES CTR, INST ENZYMOL, H-1113 BUDAPEST, HUNGARY; UNIV UTRECHT, FAC MED, DEPT CELL BIOL, NL-3584 CX UTRECHT, NETHERLANDS; UNIV UTRECHT, INST BIOMEMBRANES, NL-3584 CX UTRECHT, NETHERLANDS

COUNTRY OF AUTHOR: NETHERLANDS; HUNGARY

SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (4 AUG 2000) Vol. 275, No. 31, pp. 23530-23539.
Publisher: AMER SOC BIOCHEMISTRY MOLECULAR BIOLOGY INC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814.
ISSN: 0021-9258.

DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 51

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The human MDR3 gene is a member of the multidrug resistance (MDR) gene family. The MDR3 **P-glycoprotein** is a transmembrane protein that translocates phosphatidylcholine. The **MDR1** **P-glycoprotein** related transports cytotoxic drugs. Its overexpression can make cells resistant to a variety of drugs. Attempts to show that MDR3 **P-glycoprotein** can cause MDR have been unsuccessful thus far. Here, we report an increased directional transport of several **MDR1** **P-glycoprotein** substrates, such as digoxin, paclitaxel, and vinblastine, through polarized monolayers of MDR3-transfected cells. Transport of other good **MDR1** **P-glycoprotein** substrates, including cyclosporin A and dexamethasone, was not detectably increased. MDR3 **P-glycoprotein**-dependent transport of a short-chain phosphatidylcholine analog and drugs was inhibited by several MDR reversal agents and other drugs, indicating an interaction between these compounds and MDR3 P-gp, Insect cell membranes from Sf9 cells overexpressing MDR3 showed specific MgATP binding and a vanadate-dependent, N-ethylmaleimide-sensitive nucleotide trapping activity, visualized by covalent binding with [alpha-P-32]8 azido-ATP. Nucleotide trapping was (nearly) abolished by paclitaxel, vinblastine, and the MDR reversal agents verapamil, cyclosporin A, and PSC 833. We conclude that MDR3 **P-glycoprotein** can bind and transport a subset of **MDR1** **P-glycoprotein** substrates. The rate of MDR3 P-wglycoprotein-mediated transport is low for most drugs, explaining why this protein is not detectably involved in multidrug resistance. It remains possible, however, that drug binding to MDR3 **P-glycoprotein** could adversely affect phospholipid or toxin secretion under conditions of stress (e.g. in pregnant heterozygotes with one MDR3 null **allele**).

L4 ANSWER 7 OF 15 SCISEARCH COPYRIGHT 2002 ISI (R)

ACCESSION NUMBER: 1999:658128 SCISEARCH

THE GENUINE ARTICLE: 228GM

TITLE: Species differences in the transport activity for organic anions across the bile canalicular membrane

AUTHOR: Ishizuka H (Reprint); Konno K; Shiina T; Naganuma H; Nishimura K; Ito K; Suzuki H; Sugiyama Y

CORPORATE SOURCE: SANKYO CO LTD, ANALYT & METAB RES LABS, SHINAGAWA KU, 2-58 HIROMACHI 1 CHOME, TOKYO, JAPAN (Reprint); SANKYO CO LTD, BIOMED RES LABS, SHINAGAWA KU, TOKYO, JAPAN; UNIV TOKYO,

GRAD SCH PHARMACEUT SCI, TOKYO, JAPAN
COUNTRY OF AUTHOR: JAPAN
SOURCE: JOURNAL OF PHARMACOLOGY AND EXPERIMENTAL THERAPEUTICS, (SEP 1999) Vol. 290, No. 3, pp. 1324-1330.
Publisher: AMER SOC PHARMACOLOGY EXPERIMENTAL THERAPEUTICS 9650 ROCKVILLE PIKE, BETHESDA, MD 20814-3998.
ISSN: 0022-3565.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 39
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Species differences in the transport activity mediated by canalicular multispecific organic anion transporter (cMOAT) were examined using temocaprilat, an angiotensin-converting enzyme inhibitor whose biliary excretion is mediated predominantly by cMOAT,and 2,4-dinitrophenyl-S-glutathione, a typical substrate for cMOAT, in a series of in vivo and in vitro experiments. Temocaprilat was infused to examine the biliary excretion rate at steady-state. The in vivo transport clearance values across the bile canalicular membrane, defined as the biliary excretion rate divided by the hepatic unbound concentrations, were 9.8, 39.2, 9.2, 1.1, and 0.8 ml/min/kg for mouse, rat, guinea pig, rabbit, and **dog** , respectively. The K-m and V-max values for ATP-dependent uptake of 2,4-dinitrophenyl-S-glutathione into canalicular membrane vesicles were 15.0, 29.6, 16.1, 55.8, and 30.0 mu M and 0.38, 1.90, 0.15, 0.47, and 0.23 nmol/min/mg protein, yielding the in vitro transport clearance across the bile canalicular membrane (V-max/K-m) of 25.5, 64.2, 9.4, 8.4, and 7.7 for mouse, rat, guinea pig, rabbit, and **dog**, respectively. A close in vivo and in vitro correlation was observed among animal species for the transport clearance across the bile canalicular membrane. These results suggest that the uptake experiments with canalicular membrane vesicles can be used to quantitatively predict in vivo excretion across the bile canalicular membrane.

L4 ANSWER 8 OF 15 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 1998:915287 SCISEARCH
THE GENUINE ARTICLE: 142NR
TITLE: Functional multidrug resistance protein (MRP1) lacking the N-terminal transmembrane domain
AUTHOR: Bakos E; Evers R; Szakacs G; Tusnady G E; Welker E; Szabo K; deHaas M; vanDeemter L; Borst P; Varadi A; Sarkadi B (Reprint)
CORPORATE SOURCE: HUNGARIAN ACAD SCI, NATL INST HAEMATOL & IMMUNOL, RES GRP, DAROCZI U 24, H-1113 BUDAPEST, HUNGARY (Reprint); HUNGARIAN ACAD SCI, NATL INST HAEMATOL & IMMUNOL, RES GRP, H-1113 BUDAPEST, HUNGARY; HUNGARIAN ACAD SCI, BIOL RES CTR, INST ENZYMOL, H-1051 BUDAPEST, HUNGARY; NETHERLANDS CANC INST, DIV MOL BIOL, NL-1066 CX AMSTERDAM, NETHERLANDS
COUNTRY OF AUTHOR: HUNGARY; NETHERLANDS
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (27 NOV 1998) Vol. 273, No. 48, pp. 32167-32175.
Publisher: AMER SOC BIOCHEMISTRY MOLECULAR BIOLOGY INC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814.
ISSN: 0021-9258.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 32
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The human multidrug resistance protein (MRP1) causes drug resistance by extruding drugs from tumor cells. In addition to an MDR-like core, MRP1 contains an N-terminal membrane-bound region (TMD0) connected to the core by a cytoplasmic linker (L-0). We have studied truncated MRP1 versions containing either the MDR-like core alone or the core plus linker L-0,

produced in the baculovirus-insect (Sf9) cell system. Their function was examined in isolated membrane vesicles. Full-length MRP1 showed ATP-dependent, vanadate-sensitive accumulation of leukotriene C-4 and N-ethylmaleimide glutathione. In addition, leukotriene C-4-stimulated, vanadate-dependent nucleotide occlusion was detected. The MDR-like core was virtually inactive. Co-expression of the core with the N-terminal region including L-0 fully restored MRP1 function. Unexpectedly, a truncated MRP1 **mutant** lacking the entire TMD, region but still containing L-0 behaved like wild-type MRP1 in vesicle uptake and nucleotide trapping experiments. We also expressed the MRP1 constructs in polarized **canine** kidney derived MDCKII cells. Like wild-type MRP1, the MRP1 protein without the TMD, region was routed to the lateral plasma membrane and transported dinitrophenyl glutathione and daunorubicin, The TMD0L0 and the MRP1 minus TMD0L0 remained in an intracellular compartment. Taken together, these experiments strongly suggest that the TMD0 region is neither required for the transport function of MRP1 nor for its proper routing to the plasma membrane.

L4 ANSWER 9 OF 15 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 1998:71833 SCISEARCH
THE GENUINE ARTICLE: YQ827
TITLE: Mutations of the p53 gene in **canine** lymphoma and evidence for germ line p53 mutations in the **dog**
AUTHOR: Veldhoen N (Reprint); Stewart J; Brown R; Milner J
CORPORATE SOURCE: UNIV YORK, DEPT BIOL, YCRC RES GRP P53, YORK YO1 5DD, N YORKSHIRE, ENGLAND (Reprint); UNIV GLASGOW, CRC, BEATSON LABS, DEPT MED ONCOL, GLASGOW G61 1BD, LANARK, SCOTLAND
COUNTRY OF AUTHOR: ENGLAND; SCOTLAND
SOURCE: ONCOGENE, (15 JAN 1998) Vol. 16, No. 2, pp. 249-255. Publisher: STOCKTON PRESS, HOUNDMILLS, BASINGSTOKE, HAMPSHIRE, ENGLAND RG21 6XS.
ISSN: 0950-9232.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 49
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Mutations of the p53 gene are associated with a number of non-lymphoid cancers of the **dog**. The present study investigates the p53 gene status within **canine** patients treated for primary and secondary lymphoma. Three out of eight patients exhibited p53 gene mutations, These included one patient with a germ line mutation and two patients with de novo p53 mutations associated with the secondary lymphoma. **Allelic** loss of the p53 gene was also observed within primary and secondary tumours of the three **canine** patients. The results indicate that germ line p53 mutations exist in **dogs** and may be involved in the known predisposition of some breeds to cancer, The presence of therapy-related p53 point mutations was found to be associated with chemoresistant secondary lymphomas. A causative role for DNA-damaging chemotherapy in de novo mutation of the p53 gene is discussed, Characterization of p53 inactivation in **canine** tumorigenesis may provide a valuable clinical model for assessing the efficacy and optimal therapeutic regimens of anti-cancer agents.

L4 ANSWER 10 OF 15 MEDLINE
ACCESSION NUMBER: 1998086200 MEDLINE
DOCUMENT NUMBER: 98086200 PubMed ID: 9426231
TITLE: Transport of glutathione prostaglandin A conjugates by the multidrug resistance protein 1.
AUTHOR: Evers R; Cnubben N H; Wijnholds J; van Deemter L; van Bladeren P J; Borst P
CORPORATE SOURCE: Division of Molecular Biology, The Netherlands Cancer Institute, Amsterdam.
SOURCE: FEBS LETTERS, (1997 Dec 8) 419 (1) 112-6.

Journal code: 0155157. ISSN: 0014-5793.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199801
ENTRY DATE: Entered STN: 19980206
Last Updated on STN: 19980206
Entered Medline: 19980126

AB The human multidrug resistance protein MRP1 mediates transport of organic substrates conjugated to glutathione, glucuronide, or sulfate. The naturally occurring prostaglandins A1 and A2 can form two diastereomeric glutathione S-conjugates, and it has been speculated that these might be substrates for MRP1. Here we present evidence that polarized MDCKII cells expressing MRP1 cDNA transport PGA1-GS to the basolateral side of a cell monolayer, in accordance with the lateral localization of human MRP1 in these cells. Furthermore, we show that vesicles made from yeast cells expressing MRP1 cDNA and from mouse erythrocytes (known to contain mrp1) actively accumulate both diastereomers of PGA2-GS with a similar efficiency. Recently, we generated mice with a homozygous **mutant** mrp1 **allele**. Uptake of PGA2-GS in vesicles made from erythrocytes of these mice was 3.2 times lower than in wild-type vesicles, but was still significantly above background. This residual transport activity was partly inhibited by methotrexate and cAMP, whereas mrp1-mediated activity was unaffected by these compounds. We conclude that mouse erythrocytes contain at least two transport systems for PGA2-GS. One of these is mrp1; the other one has not been identified yet, but can be inhibited by methotrexate and cAMP.

L4 ANSWER 11 OF 15 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:736020 CAPLUS
DOCUMENT NUMBER: 137:268387
TITLE: Rhesus monkey **P-glycoproteins** and uses thereof
INVENTOR(S): Crespi, Charles L.; Hanscom, Sarah R.
PATENT ASSIGNEE(S): Gentest Corp., USA
SOURCE: PCT Int. Appl., 103 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002074048	A2	20020926	WO 2002-US8325	20020319

W: AT, CA, JP
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, LU, NL, PT, SE, TR
PRIORITY APPLN. INFO.: US 2001-277095P P 20010319

AB The invention pertains to rhesus monkey **P-glycoproteins** and related **P-glycoproteins** which include rhesus-specific amino acids, as well as nucleic acids which encode those polypeptides. The present invention also includes fragments and biol. functional **variants** of the rhesus monkey **P-glycoprotein**. The invention further relates to methods of using such rhesus monkey **P-glycoprotein** nucleic acids and polypeptides, esp. in methods for detg. bioavailability of drugs and for screening for inhibitors of rhesus **PGP**. Also included are rhesus **PGP** inhibitors which inhibit rhesus **PGP** activity by inhibiting the expression or function of rhesus **PGP**.

L4 ANSWER 12 OF 15 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:555699 CAPLUS
DOCUMENT NUMBER: 137:105497

TITLE: Truncation mutation of **mdr1 variants** associated with ivermectin sensibility detected in **dog** and methods for their use in diagnosis
INVENTOR(S): Mealey, Katrina L.; Bentjen, Steven A.
PATENT ASSIGNEE(S): Washington State University Research Foundation, USA
SOURCE: PCT Int. Appl., 50 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002057499	A2	20020725	WO 2002-US868	20020110
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2002177147	A1	20021128	US 2002-44671	20020110

PRIORITY APPLN. INFO.: US 2001-261578P P 20010112
US 2001-314829P P 20010824

AB This invention provides the identification of a truncation polymorphism of the **mdr1** gene that is linked to ivermectin sensitivity in subjects, such as collies. Specifically, a four base pair deletion is detected at the position of 294-297 of **canine mdr1** gene cDNA affecting the codon for amino acid residue of 75 and causing a frame shift and truncation at residue 91 and 111. This resulted truncation products of P-gp causes sensitivity to ivermectin and other drugs that serve as P-gp substrates. Furthermore, the frequency of the **MDR1** gene mutation (polymorphism) assocd. with ivermectin sensitivity in a sample population of Collies in Washington and Idaho. A high percentage of a sample population of Collies in Washington and Idaho are affected or carriers of the **mutant MDR1 allele** assocd. with ivermectin sensitivity. A similar frequency of this mutation may be detected in **dogs** from other geog. areas. Pharmacol. treatment with ivermectin, loperamide, vincristine, and other drugs that are substrates of **P-glycoprotein**, the **MDR1** gene product, may result in neurol. toxicosis in a high percentage of Collies. Also provided are methods for detecting drug transport sensibility in a subject, and animal models and in vitro cell systems using cells from animals having an **mdr1** truncation.

L4 ANSWER 13 OF 15 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:265564 CAPLUS
DOCUMENT NUMBER: 134:290384
TITLE: Compositions and methods for modulating ATP-binding cassette transmembrane reporter protein expression and identification of therapeutic agents
INVENTOR(S): Hamilton, Joshua W.; Stanton, Bruce A.
PATENT ASSIGNEE(S): Trustees of Dartmouth College, USA
SOURCE: PCT Int. Appl., 27 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001025400	A2	20010412	WO 2000-US27443	20001004
WO 2001025400	A3	20010830		

W: CA, JP, US
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE

PRIORITY APPLN. INFO.: US 1999-158000P P 19991006
US 2000-194274P P 20000403

AB Methods and compns. for modulating cell surface protein expression are provided. The compns. of the present invention are gene constructs comprising ATP-binding cassette transmembrane reporter proteins. The effects of doxorubicin (Dox) on .DELTA.F508 CFTR expression were examd. in a **canine** kidney MDCK-derived cell line that had been stably transfected with a human .DELTA.F508 CFTR cDNA construct expressed under the control of a CMV promoter. Dox had no effect on chloride currents in either controls. However, Dox statistically significantly increased chloride currents in the cell line expressing the **mutant** CFTR by approx. 1.7-fold.

L4 ANSWER 14 OF 15 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:247498 CAPLUS
DOCUMENT NUMBER: 134:276530
TITLE: Cloning and characterization of **P-glycoproteins** from Macaca fascicularis and uses for drug bioavailability or drug screening studies
INVENTOR(S): Stocker, Penny J.; Steimel-Crespi, Dorothy T.; Crespi, Charles L.
PATENT ASSIGNEE(S): Gentest Corporation, USA
SOURCE: PCT Int. Appl., 84 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001023565	A1	20010405	WO 2000-US26592	20000928
EP 1220917	A1	20020710	EP 2000-968443	20000928

W: AU, CA, JP
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY

PRIORITY APPLN. INFO.: US 1999-156921P P 19990928
US 1999-158818P P 19991012
WO 2000-US26592 W 20000928

AB The invention pertains to cynomologous monkey **P-glycoproteins** (multidrug transporter **MDR1**) and related **P-glycoproteins** which include cynomologous-specific amino acids, as well as nucleic acids which encode those polypeptides. The cDNA and encoded amino acid sequences of the cynomologous monkey **P-glycoprotein** and its **allelic variant** are disclosed. The present invention also includes fragments and biol. functional **variants** of the cynomologous monkey **P-glycoprotein**. The invention further relates to methods of using such cynomologous monkey **P-glycoprotein** nucleic acids and polypeptides, esp. in methods for detg. bioavailability of drugs and for screening for inhibitors of cynomologous **PGP**. Also included are cynomologous **PGP** inhibitors which inhibit cynomologous **PGP** activity by inhibiting the expression or function of cynomologous **PGP**.

REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 15 OF 15 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:247473 CAPLUS
DOCUMENT NUMBER: 134:276524
TITLE: Cloning and characterization of **dog** **P-glycoproteins** and uses for drug bioavailability or drug screening
INVENTOR(S): Stocker, Penny J.; Steimel-Crespi, Dorothy T.; Crespi, Charles L.; Reif, Timothy C.; Patten, Christopher J.
PATENT ASSIGNEE(S): Gentest Corporation, USA
SOURCE: PCT Int. Appl., 111 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001023540	A2	20010405	WO 2000-US26767	20000928
WO 2001023540	A3	20011018		
W: AU, CA, JP				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
EP 1220911	A2	20020710	EP 2000-967072	20000928
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL				

PRIORITY APPLN. INFO.: US 1999-156510P P 19990928
WO 2000-US26767 W 20000928

AB The invention pertains to **dog P-glycoprotein** (multidrug transporter **MDR1**) and related **P-glycoproteins** which include **dog**-specific amino acids, as well as nucleic acids which encode those polypeptides. The cDNA and encoded amino acid sequences of the **dog P-glycoprotein** and its **allelic variants** are disclosed. The present invention also includes fragments and biol. functional **variants** of the **dog P-glycoprotein**. The invention further relates to methods of using such **dog P-glycoprotein** nucleic acids and polypeptides, esp. in methods for detg. bioavailability of drugs and for screening for inhibitors of **dog PGP**. Also included are **dog PGP** inhibitors which inhibit **dog PGP** activity by inhibiting the expression or function of **dog PGP**.